

An Empirical Comparison of Lead Exposure Pathway Models

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Structural equation modeling is a statistical method for partitioning the variance in a set of interrelated multivariate outcomes into that which is due to direct, indirect, and covariate (exogenous) effects. Despite this model's flexibility to handle different experimental designs, postulation of a causal chain among the endogenous variables and the points of influence of the covariates is required. This has motivated the researchers at the University of Cincinnati Department of Environmental Health to be guided by a theoretical model for movement of lead from distal sources (exterior soil or dust and paint lead) to proximal sources (interior dust lead) and then finally to biologic outcomes (handwipe and blood lead). The question of whether a single structural equation model built from proximity arguments can be applied to diverse populations observed in different communities with varying lead amounts, sources, and bioavailabilities is addressed in this article. This reanalysis involved data from 1855 children less than 72 months of age enrolled in 11 studies performed over approximately 15 years. Data from children residing near former ore-processing sites were included in this reanalysis. A single model adequately fit the data from these 11 studies; however, the model needs to be flexible to include pathways that are not frequently observed. As expected, the more proximal sources of interior dust lead and handwipe lead were the most important predictors of blood lead; soil lead often had a number of indirect influences. A limited number of covariates were also isolated as usually affecting the endogenous lead variables. The blood lead levels surveyed at the ore-processing sites were comparable to and actually somewhat lower than those reported in the Third National Health and Nutrition Examination Survey. Lessened bioavailability of the lead at certain of these sites is a probable reason for this finding. — *Environ Health Perspect* 106(Suppl 6):1577–1583 (1998). <http://ehpnet1.niehs.nih.gov/docs/1998/Suppl-6/1577-1583succop/abstract.html>

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Researchers at the University of Cincinnati Department of Environmental Health have been collecting and analyzing lead source data from numerous field studies for over 15 years. The importance of proximal lead sources to increasing children's lead burden has been repeatedly demonstrated in these data. To this point, separate analyses of the data from each study site have been

performed (1–16). The question addressed in this article is what is the degree of consistency among the data analyses that have been performed.

In 1985, the statistical technique of structural equation modeling was introduced into the environmental health literature (3). This method may be used to partition the variance in a set of interrelated

outcomes between that which is caused by a direct influence (i.e., not mediated by other variables in the model) and that which is related through one or more indirect pathways (i.e., mediated by at least one other variable in the model). The structural equation approach allows researchers to postulate richer and more realistic models of environmental exposure, as not every possible predictor must be specified to have a direct influence on the final outcome (e.g., a measure of internal exposure such as children's measured blood lead).

These models are specified in a manner similar to ordinary regression models. However, because an entire causal chain of events or outcomes must be specified, additional thought must be given to the order in which these events take place (as reflected in changes/differences in the measured variables). In the models fit by researchers at the University of Cincinnati, the postulated chain of events is that of movement of lead from paint and soil to dust on floors (and/or other surfaces), to children's hands (and/or toys), and finally to their blood (Figure 1). The model shown in Figure 1 is a theoretical model that has served to guide the various statistical analyses. The direction of causality, of necessity, is imposed on the model's pathways, much as independent variables are chosen to be predictors of dependent variables, which are the outcome variables used in both a structural equation or a multiple regression analysis. Choice of the direction of causality may be made in terms of one or more considerations, including timing of events (antecedents are expected to predict future outcomes), proximity (the model is built from distal to proximal sources or biologic outcomes), and amount of contamination (a sampled media with larger amounts of lead might be expected to contaminate a sampled media that demonstrated a lesser amount of lead). The cross-sectional model shown in Figure 1 was built primarily on proximity arguments. Site-specific differences (e.g., in the bioavailability of lead, the general condition of the housing stock, the amount of groundcover, or the type of soil) also have introduced variability into the relative contributions that each of these sources makes to predicting a child's blood lead.

In addition to the pathways between the lead sources and the outcome of blood lead, covariates (i.e., other measurements made at the site that may modify the

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Abbreviations used: GM, geometric mean; mg/cm², milligram per square centimeter; µg, microgram; µg/m², microgram per square meter; µg/dl, microgram per deciliter; NHANES III, the Third National Health and Nutrition Examination Survey; ppm, parts per million.

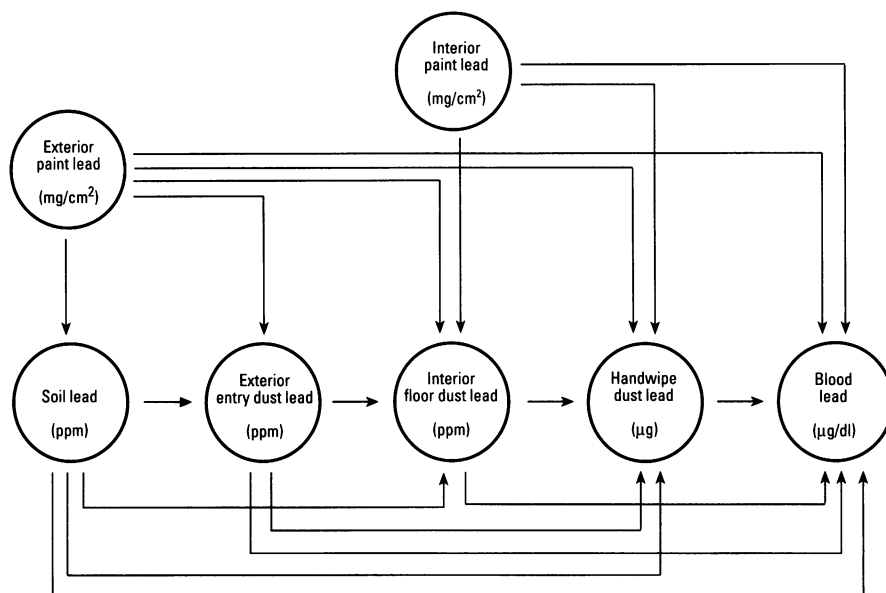


Figure 1. Generic structural equations pathway model of lead exposure.

associations shown in Figure 1) must be taken into account. Covariates that frequently are associated with children's blood lead levels include the family's socioeconomic status (income and/or education), the child's age, and the child's hand-to-mouth activity. House age and condition often are significant predictors of the lead source levels, in particular, the paint lead loading (measured in amount of lead per unit area, e.g., mg/cm²), or soil lead and dust lead loadings (measured in amount of lead per unit area, e.g., µg/m²) and concentrations (characterized as parts per million). Proportion of soil area with groundcover affects exterior dust lead levels, as well as soil lead levels in some studies. The covariates that are statistically significant are often not the same for each community, as the effects of these variables are more likely to be site- or population-specific than the endogenous pathways shown in Figure 1.

The question that is addressed in this article is the degree of similarity in the results documented from a number of different sites in which community lead studies have been undertaken. The more similar the results, the more appropriate that a single model be used for scientific and regulatory purposes and to estimate the dose-response relationships in different communities.

Methods

In this analysis, 1855 children less than 72 months of age were available from 11 studies [Bingham Creek, Utah (1); Butte, Montana (2); Cincinnati, Ohio, Lead in

Children Study (3,4); Cincinnati, Ohio, soil lead abatement study (5–7); Leadville, Colorado (8); Magna, Utah (9); Midvale, Utah (10,11); Palmerton, Pennsylvania (12); Sandy, Utah (13); Telluride, Colorado (14,15); and Trail, British Columbia (16)]. The following is a brief description of each site.

Bingham Creek, Utah, 15 miles south of Salt Lake City, is heavily contaminated with mill tailings that were transported downstream from the copper and lead mines located 10 to 15 miles to the west of the most heavily contaminated area. Transport of tailings down the creek ceased in the mid-1930s. Recently, new middle income subdivisions have been built on lead-contaminated soils along a 10-mile stretch of the Bingham Creek flood plain (1).

Butte, Montana, population 39,000, is the site of extensive deep and open-pit copper mining. Some open-pit mining continues today. Smelters ceased operation by about 1910. There are extensive lead-containing mine waste dumps and tailings ponds throughout the community. Lead in paint and drinking water pose additional concerns (2).

The Cincinnati Lead in Children Study is a longitudinal study of the effects of lead on child development in a cohort of low-income families living in the inner city. Most housing, which was built in the late 19th century, is poorly maintained and contains large quantities of lead-based paint. Extensive environmental data on

this cohort was collected between 1981 and 1986 (3,4). A cross-sectional sample of children that maximized the amount of nonmissing data and minimized the lag time between the measurement of the children's blood lead and the measurement of the lead associated with their residences was chosen for the purpose of this reanalysis.

The Cincinnati soil lead abatement project focused on lead exposure reduction in families residing in inner city apartment buildings that had been renovated and contained little or no lead-based paint. Primary sources of exposure were high levels of lead in street dust and soil in vacant lots and playgrounds (5–7). Only preintervention data collected for this study were used in this reanalysis.

Leadville, Colorado, population 3800, was home to very extensive lead mining, milling, and smelting operations. Most mines and smelters closed by 1970. One mine and mill remain in operation. Lead-based paint is a major source of exposure in over half the housing (8).

Magna, Utah, is located just south of a 6000-acre tailings pond and downwind from a very large active copper smelter about 15 miles west of Salt Lake City. Residential soils in a 15-square-block, low-income neighborhood closest to the smelter were evaluated and found to have acceptably low levels of lead. However, lead-based residential paint posed a considerable concern (9).

Midvale, Utah, population 12,000, is located about 12 miles south of Salt Lake City. It was home to a zinc/lead mill and smelter. The smelter closed in the mid-1950s and the mill ceased operation in the early 1970s. Residential soils contained high levels of zinc and lead. Homes built prior to 1950 contain high levels of lead in paint (10,11).

Palmerton, Pennsylvania, population 5400, is located about 100 miles northwest of Philadelphia. A large zinc smelter with plant sites on each side of the town operated for over 75 years in this valley. Residential soils contain high levels of lead and cadmium (12).

Sandy, Utah, historic community about 15 miles south of Salt Lake City, was the site of several lead smelters at the turn of the century. Residential soils in a 15-square-block area around these former smelters contain lead and arsenic. Housing is old but well maintained (13).

Telluride, Colorado, population 1200, was a silver and lead mining and milling community at the turn of the century.

Operations ceased in 1977. Old housing contains considerable quantities of lead-based paint (14,15).

Trail, British Columbia, population 15,000, is located on the Columbia River due north of Spokane, Washington, and is home to one of the few remaining active lead and zinc smelters in North America. High lead emissions contaminated much of the residential area in the immediate vicinity of the plant. New technology has greatly reduced emissions but residual lead dust remains a problem (16).

Data collection in these studies was performed between 1981 and 1995, with most of the studies being performed since 1989. Environmental and blood collection procedures were standardized and all studies incorporated a rigorous quality control plan. Community geometric mean

(GM) blood lead levels ranged between 2.6 µg/dl in Bingham Creek, Utah, and 12.9 µg/dl in the Cincinnati Lead in Children Study. Handwipe lead was highest in Trail, British Columbia (GM = 9.0 µg), and lowest in Bingham Creek (GM = 2.0 µg); however, handwipes were not collected in four of the studies. A complete comparison of lead exposures at the various sites is shown in Table 1. A summary of the number of elevated blood leads at various ore-processing sites is shown in Table 2. This table also includes data collected in the town of Aspen, Colorado, a site at which a small study was performed in 1996 (17). From this table, it can be seen that the blood lead levels at the ore-processing sites do not differ appreciably from that of children nationally (according to the the Third National

Health and Nutrition Examination Survey (NHANES III) National Blood Lead Survey (18), and actually are somewhat lower than the national average. Lessened bioavailability of the lead at certain of these sites is a probable reason for this finding.

The generic model for pathways from lead sources to children's blood leads (Figure 1) was applied to data from each of the 11 studies as closely as possible. Not all data were collected at all sites (e.g., handwipe lead), and the questionnaires used for collecting much of the covariate information did not become completely standardized until the later field studies undertaken in the western United States. Thus, some variables were not measured at certain sites.

In the analyses of each community's data, a strategy of backward elimination of

Table 1. Comparison of lead exposure at study sites (children < 72 months of age).

Variable ^a	Unit	Cincinnati child study (3,4) 1981–1985	Cincinnati soil lead study (5–7) 1989–1991	Telluride, CO (14,15) 1987	Midvale, UT (10,11) 1989	Butte, MT (2) 1990	Leadville, CO (8) 1991	Trail, B.C. (16) 1992	Bingham Creek, UT (1) 1993	Magna, UT (9) 1994	Sandy, UT (13) 1994	Palmerton, PA (12) 1994
Blood lead concentration	µg/dl	12.9	10.5	6.1	5.2	3.5	4.8	11.0	2.6	4.0	3.0	4.7
Handwipe lead	µg	7.0	6.0	4.5	—	—	—	9.0	2.0	4.0	3.0	—
Interior dust lead concentration	ppm	1548	344	281	438	451	638	1014	110	178	212	589
Interior dust lead loading	µg/m ²	976	75	—	161	71	195	280	31	116	80	122
Interior dust loading	g/m ²	—	—	—	0.44	0.14	0.31	—	0.28	0.58	0.36	0.23
Exterior entry dust lead concentration	ppm	1830	1376	178	466	671	797	2427	72	220	260	1103
Street dust lead concentration	ppm	—	1108	—	—	242	877	920	10.3	81	204	—
Perimeter soil lead concentration	ppm	—	—	145	342	472	808	620	57	—	367	780
Sandbox/play area lead concentration	ppm	—	—	—	78	201	244	—	38	—	220	—
Garden soil lead concentration	ppm	—	—	—	295	372	464	—	53	—	362	—
Bare yard area lead concentration	ppm	—	—	—	313	365	774	—	67	—	344	—
Maximum exterior paint lead loading	mg/cm ²	4.9	1.2	1.9	1.2	5.0	1.6	—	<0.35	2.7	2.3	6.2
Maximum interior paint lead loading	mg/cm ²	2.2	0.9	2.6	1.0	2.5	1.5	—	<0.35	1.6	1.7	<0.35
Water lead concentration	ppm	—	1.6	1.2	1.4	6.1	1.0	—	1.2	1.0	5.0	—
When housing was constructed	Year	1890–1940	1890–1940	—	1890–1940	1914	1957	—	1984	1928	1961	1921
Child's age	Months	15.1	31.0	37.9	33.8	29.2	38.4	38.5	38.2	35.9	38.0	38.6
Mouthing behavior summary score	^b	—	—	—	14	33	26	14	25	25	25	23
Residency length	Months	9	17	—	12	27	24	—	34	31	27	30
Socioeconomic status	^c	17	17	46	26	31	30	—	40	27	32	32

—, no data exists. ^aTabled values are geometric means except for "When housing was constructed," whose values are arithmetic mean year of construction, where available, or the modal epoch of construction if not (i.e., 1890–1940); and "Child's age," "Mouthing," "Residency length," and "Socioeconomic status," whose tabled values are arithmetic means. ^bPossible range = 10 (low score) to 40 (high score); constructed from 10 items each measured on a 4-point scale. ^cPossible range = 8 (low score) to 66 (high score).

Table 2. Summary of elevated blood lead levels of children < 72 months at former ore-processing sites.^a

Study site	Year	Number screened	Number > 10 µg/dl	% > 10 µg/dl	Number > 20 µg/dl	% > 20 µg/dl
Telluride, CO ^b	1986	94	20	21.3	0	0.0
Leadville, CO ^c	1987	150	61	40.0	14	9.3
Midvale, UT ^b	1989	181	23	12.7	1	0.6
Butte, MT ^b	1990	294	15	5.1	2	0.7
Leadville, CO ^b	1991	315	26	8.3	0	0.0
Palmerton, PA ^c	1991	84	19	22.6	0	0.0
Bingham Creek, UT ^b	1993	971	8	0.8	1	0.1
Sandy, UT ^b	1993–1994	129	0	0.0	0	0.0
Sandy, UT ^d	1995	257	0	0.0	0	0.0
Palmerton, PA ^b	1994	140	9	6.4	1	0.7
Magna, UT ^b	1994	162	10	6.2	0	0.0
Magna, UT ^d	1995	198	2	1.0	0	0.0
Aspen, CO ^b (17)	1996	9	0	0.0	0	0.0
Total ^e	1986–1996	2074	156	7.5	18	0.9
Total ^f	1986–1996	2459	103	4.2	5	0.2

^aThe NHANES III Blood Lead Survey (1989–1991) found that 8.9% of U.S. children under 72 months of age have blood lead levels > 10 µg/dl and 1.1% have levels > 20 µg/dl.

^bStudy conducted by the University of Cincinnati. ^cStudy conducted by the Agency for Toxic Substances and Disease Registry. ^dStudy conducted by the city/county health department. ^eThe total is based on the earlier assessment of each community. ^fThe total is based on the more recent assessment of each community.

insignificant pathways was employed. An initial model was specified, similar to that shown in Figure 1, that included all possible endogenous pathways and covariates and interaction variables postulated to affect one or more variables in the causal chain. The effects of insignificant interactions, i.e., cross-products formed by multiplying two or more explanatory variables in a given equation, were removed before the simple main effects from which they were formed. Insignificant effects were removed until all the remaining predictors in each structural equation were significant. At this stage, a single forward inclusion step was performed that allowed previously removed variables the opportunity to re-enter the structural equation. A final model was obtained when all effects in the model were significant and all excluded predictors were insignificant, as based on a test for inclusion in the final model. In all of these analyses, blood lead and environmental lead data were transformed to their natural logarithm to normalize the statistical distributions. An alpha level of 0.05 was used to judge statistical significance for each postulated pathway in each study.

Results

A summary of the significant pathways among the endogenous variables from structural equation models fit separately to each site's data is shown in Table 3. Exterior paint lead loading was found to be a significant predictor of soil lead concentration in 3 of 7 studies (43%) in which this pathway was tested. The concentration of entry dust lead was predicted significantly in 3 of 8 studies (37.5%) by exterior paint lead loading, in 1 of 8 studies

(12.5%) by interior paint lead loading, and in 6 of 7 (86%) studies by soil lead concentration. Concentration of floor dust lead was significantly related to exterior paint lead loading in 2 of 9 studies (22%), to interior paint lead loading in 4 of 10 studies (40%), to soil lead concentration in 6 of 8 studies (75%), and to entry dust lead concentration in 7 of 10 studies (70%). Handwipe lead was predicted by interior paint lead loading in 2 of 6 studies (33%), by soil lead concentration in 2 of 3 studies (67%), by entry dust lead concentration in 1 of 6 studies (17%), and by floor dust lead concentration in 5 of 6 studies (83%). Finally, exterior paint lead loading was found to be a significant factor in explaining the variance in blood lead in 2 of 9 studies (22%). Interior paint lead loading contributed significantly in 1 of 10 studies (10%), soil lead concentration in 3 of 8 studies (37.5%), entry dust lead concentration in 1 of 10 studies (10%), floor dust lead concentration in 7 of 11 studies (64%), and handwipe lead in 4 of 6 studies (67%). These results are also shown in Figure 2, which depicts the relative importance of each of the postulated pathways by arrows of various widths (no arrow is shown for a pathway that was never found to be significant).

The results shown in Table 3 and Figure 2 address the extent to which the various pathways postulated to exist by the theoretical structural equation model can be generalized to other sites. The strength of the individual pathways to affect blood lead can be demonstrated by estimating the simple regression relationship between the geometric mean blood lead and the geometric mean environmental lead, as observed from

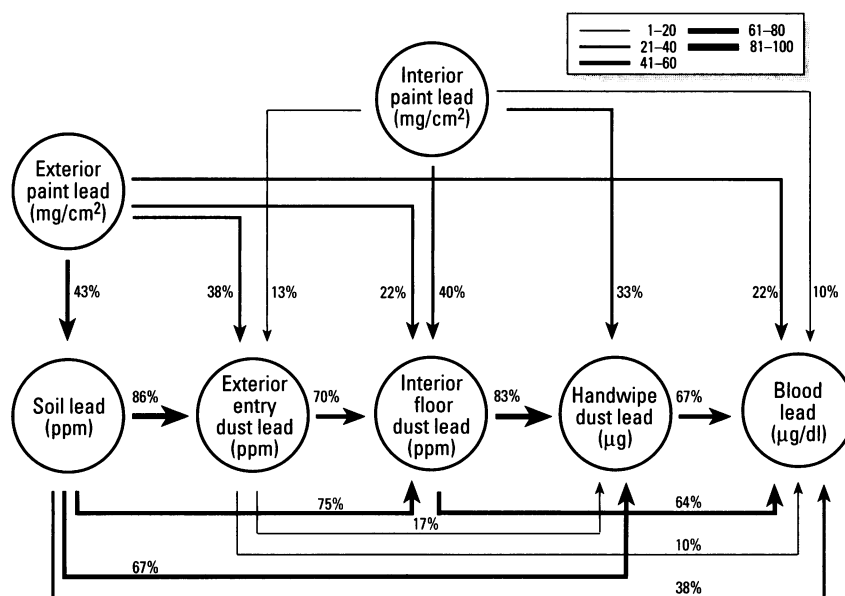
the data collected within each study. The results of this approach are summarized in Table 4. The strongest relationships were observed between blood lead and interior floor dust lead loading ($R^2 = 0.96$, after excluding the Cincinnati, Ohio, soil lead abatement project and Trail, British Columbia, which were outliers) or handwipe lead ($R^2 = 0.90$); the weakest were between blood lead and exterior or interior paint lead ($R^2 = 0.07$). The regression models also predict that increasing exposure to lead is associated with elevated blood lead levels. If these models indeed reflect causal relationships, then diminishing the amount of lead in the environment will result in a lesser lead burden for children who, in the future, reside in these environments. The model for estimating blood lead from handwipe lead predicts that children whose handwipe lead differs by about 9 µg (from 10–1 µg) have a reduced blood lead of about 14 µg/dl (from approximately 15–1 µg/dl). Somewhat smaller reductions in blood lead would be expected for differences in dust lead or soil lead concentrations of 1000 ppm, from 1100 to 100 ppm (ranging between 6.5 µg/dl for dust and 2.2 µg/dl for soil); for differences in floor dust lead loadings of about 1000 µg/m², from 1100 to 100 µg/m² (approximately a 9.0 µg/dl decrease); or for a difference in paint lead of about 2.5 mg/cm², from 3.0 to 0.5 mg/cm² (ranging between 2.1 µg/dl for interior paint lead and 1.3 µg/dl for exterior paint lead).

For comparison, a linear regression model was estimated for predicting each site's arithmetic mean blood lead from the arithmetic mean environmental lead. These results are shown in Table 5. The fit of the

Table 3. Summary of significant direct pathways among the endogenous variables.^a

Pathways	Cincinnati child study <i>n</i> = 149	Telluride, CO <i>n</i> = 45	Cincinnati soil lead study <i>n</i> = 63	Midvale, UT <i>n</i> = 112	Butte, MT <i>n</i> = 192	Leadville, CO <i>n</i> = 185	Trail, BC <i>n</i> = 180	Bingham Creek, UT <i>n</i> = 657	Magna, UT <i>n</i> = 91	Sandy, UT <i>n</i> = 89	Palmerton, PA <i>n</i> = 92	Fraction of models with pathway present (%)
Soil lead												
Exterior paint	NT	0	NT	+(0.0002)	+(0.0001)	0	NT	0	NT	0	+(0.0285)	3/7 (43)
Interior paint	NT	0	NT	0	0	0	NT	0	NT	0	0	0/7 (0)
Entry dust lead												
Exterior paint	NT	+(0.0041)	0	0	NT	0	NT	0	0	+(0.0020)	+(0.0130)	3/8 (38)
Interior paint	NT	0	0	0	NT	+(0.0001)	NT	0	0	0	0	1/8 (13)
Soil lead	NT	0	NT	+(0.0001)	NT	+(0.0001)	+(0.0001)	+(0.0001)	NT	+(0.0001)	+(0.0032)	6/7 (86)
Floor dust lead												
Exterior paint	NT	0	0	+(0.0600) ^b	0	0	NT	0	0	0	+(0.0001)	2/9 (22)
Interior paint	+(0.0500)	0	0	0	0	+(0.0246)	NT	0	+(0.0033)	+(0.0009)	0	4/10 (40)
Soil lead	NT	0	NT	+(0.0001)	+(0.0001)	+(0.0024)	+(0.0024)	+(0.0001)	NT	+(0.0001)	0	6/8 (75)
Entry dust lead	+(0.0003)	+(0.0001)	+(0.0001)	0	NT	+(0.0001)	0	+(0.0001)	+(0.0001)	0	+(0.0005)	7/10 (70)
Handwipe lead												
Exterior paint	NT	0	0	NT	NT	NT	NT	0	0	0	NT	0/5 (0)
Interior paint	0	0	0	NT	NT	NT	NT	+(0.0124)	+(0.0190)	0	NT	2/6 (33)
Soil lead	NT	0	NT	NT	NT	NT	NT	+(0.0499)	NT	+(0.0001)	NT	2/3 (67)
Entry dust lead	0	0	+(0.0130)	NT	NT	NT	NT	0	0	0	NT	1/6 (17)
Floor dust lead	+(0.0082)	+(0.0072)	+(0.0001)	NT	NT	NT	NT	+(0.0500)	+(0.0001)	0	NT	5/6 (83)
Blood lead												
Exterior paint	NT	0	0	+(0.0400)	0	0	NT	0	+(0.0022)	0	0	2/9 (22)
Interior paint	0	0	0	0	0	0	NT	0	+(0.0347)	0	0	1/10 (10)
Soil lead	NT	0	NT	+(0.0200)	0	0	+(0.0001)	0	NT	+(0.0270)	0	3/8 (38)
Entry dust lead	0	0	0	0	NT	0	0	+(0.0245)	0	0	0	1/10 (10)
Floor dust lead	+(0.0086)	+(0.0119)	0	0	+(0.0001)	+(0.0006)	0	+(0.0005)	+(0.0293)	0	+(0.0261)	7/11 (64)
Handwipe lead	+(0.0016)	+(0.0062)	+(0.0001)	NT	NT	NT	NT	+(0.0001)	0	0	NT	4/6 (67)

NT, not tested; 0, tested but not significant. ^aSignificant *p* values are in parentheses; the leading + signifies the direction of the effect (positive). ^bSignificant as an interaction with paint removal.

**Figure 2.** Composite structural equations pathway model of lead exposure.

linear model to the data was generally somewhat poorer, with *R*²s varying between 0.02 for exterior paint lead and 0.90 for interior dust lead loading. The linear model also predicts a smaller decline in

blood leads over the stated ranges. For example, the expected change in blood lead because of a change in interior dust loading from 1100 to 100 µg/m² is less than 1 µg/dl given the specification of a linear model.

Discussion

Despite variability among the sites in terms of lead bioavailability, type of site, and population sampled, origin of the lead currently found in the environment, and the exact study instruments used, a great deal of commonality is found among the results in these 11 studies. With some exceptions, soil lead and paint lead were found to be indirect influences on blood lead, usually operating through a dust lead or through dust on the children's hands. Soil lead had numerous important indirect influences in these studies. However, these studies confirmed that the proximal influences of handwipe lead and interior floor dust lead tended to be the most important direct contributors to the blood lead of children under 6 years of age.

This analysis confirms that enough commonality exists among the studies and the sampled populations that a single structural equation model is probably adequate. However, the model needs to be flexible in order to account for pathways that are infrequently observed to participate in the causal chain to elevated blood lead. For example, exterior paint, interior paint, soil lead, and entry dust lead directly affected

Table 4. Meta-analysis of the relationship between log-transformed blood lead and various environmental lead sources.^a

Independent variable	Units	Intercept	Slope estimate	Squared correlation	No. of studies	Predicted decline in blood lead ^b
<i>ln</i> (handwipe lead)	μg	0.009	1.184	0.90	6	14.4
<i>ln</i> (interior dust lead loading)	μg/m ²	-0.479	0.444	0.55	10	9.1
<i>ln</i> (interior dust lead loading) ^c	μg/m ²	-0.782	0.474	0.96	8	8.6
<i>ln</i> (interior dust lead concentration)	ppm	-1.502	0.529	0.58	10	6.5
<i>ln</i> (exterior entry dust lead concentration)	ppm	-1.101	0.435	0.72	10	4.5
<i>ln</i> (perimeter soil lead concentration)	ppm	-0.015	0.233	0.65	6	2.2
<i>ln</i> (maximum interior paint lead loading)	mg/cm ²	1.562	0.232	0.07	8	2.1
<i>ln</i> (maximum exterior paint lead loading)	mg/cm ²	1.502	0.152	0.07	9	1.3

^aThese are simple relationships unadjusted for covariates. ^bPredicted decline in blood lead for a reduction in hand lead of 10–1 μg; dust lead loading of 1100 to 100 μg/m²; dust lead or soil lead concentration of 1100–100 ppm; or paint lead loading of 3.0–0.5 mg/cm², as calculated from the fitted linear regression equation: *ln*(blood lead) = intercept + slope × *ln*(environmental lead). ^cExcluding the Trail and Cincinnati soil project studies, which appear to be outliers. The exposure in these two studies appears to be primarily from exterior dust lead.

Table 5. Meta-analysis of the relationship between blood lead and various environmental lead sources.^a

Independent variable	Units	Intercept	Slope estimate	Squared correlation	No. of studies	Predicted decline in blood lead ^b
Handwipe lead	μg	1.884	0.580	0.69	6	5.2
Interior dust lead loading	μg/m ²	5.760	0.0009	0.54	10	0.9
Interior dust lead loading ^c	μg/m ²	4.588	0.0009	0.90	8	0.9
Interior dust lead concentration	ppm	4.678	0.002	0.61	10	2.4
Exterior entry dust lead concentration	ppm	3.407	0.002	0.86	10	2.3
Perimeter soil lead concentration	ppm	3.482	0.001	0.41	6	1.5
Maximum interior paint lead loading	mg/cm ²	4.821	0.488	0.09	8	1.2
Maximum exterior paint lead loading	mg/cm ²	5.557	0.227	0.02	9	0.6

^aThese are simple relationships unadjusted for covariates. ^bPredicted decline in blood lead for a reduction in hand lead of 10–1 μg; dust lead loading of 1100–100 μg/m²; dust lead or soil lead concentration of 1100–100 ppm; or paint lead loading of 3.0–0.5 mg/cm², as calculated from the fitted linear regression equation: blood lead = intercept + slope × environmental lead. ^cExcluding the Trail and Cincinnati soil project studies, which appear to be outliers. The exposure in these two studies appears to be primarily from exterior dust lead.

blood lead in the studies done in Utah, where the bioavailability of the lead in the environment may have been low or the lead in the proximal environment did not tend to break down into smaller particles (and thus become part of normal house dust). Infrequently observed effects (representing either endogenous lead source variables or the covariates of lead exposure) must be ruled out on a site-by-site basis. The effects of covariates are likely to be site or population specific. A list of the covariates that have been most frequently observed to affect each endogenous variable in the structural equation model's causal chain is provided in Table 6. The individual reports provide the estimates of the effects on blood lead and environmental lead of each of the covariates studied at the various sites.

A simple regression model performed on the various studies' blood lead and environmental lead geometric means indicated that reducing handwipe lead and dust lead levels may result in significant declines in the blood lead of children who come to reside in these environments in the future. Reducing a child's handwipe lead level from 10 to 1 μg results in an expected decrease in a young future occupant's blood lead of about 14 μg/dl. It must be pointed out that these models are not corrected for covariates or the other measured lead

Table 6. Covariates frequently found to modify the endogenous lead levels.

Endogenous outcome variable	Modifying covariate
Exterior paint lead	House age/condition
Interior paint lead	House age/condition
Soil lead	House age/condition Exterior paint condition
Exterior entry dust lead	House age/condition Exterior paint condition Percent bare soil in yard
Interior floor dust lead	House age/condition Home renovation Parental occupation Lead-related hobbies Presence of pets (dog or cat)
Handwipe lead	Child's age Parental occupation Lead-related hobbies Presence of pets (dog or cat) Percent bare soil in yard
Blood lead	Child's age Mouthing frequency Hematocrit Socioeconomic status Duration of residency Time spent outdoors

sources and therefore are most useful in demonstrating the relative expected declines, not the absolute declines that might be obtained in reducing a single source of lead. Lead in the environment presents a multimedia multisource problem

that is unlikely to be resolved by abating only one source or media in a community. A comparable linear model for each site's arithmetic means provided a somewhat poorer fit to the data and also predicted a smaller decline in low-level blood leads.

In summary, the modeling technique used to fit the data collected in these studies provides an empirical method for predicting community lead exposure levels and the inherent variability about these levels. The accuracy of these predictions is crucial, not only because of scientific interest and medical concern, but also because such expectations often serve as the basis for regulation of lead levels in the environment.

Conclusions

The statistical technique of structural equation analysis is an extremely flexible method that may be used to model environmental source data linkages with human population exposure. Although a good deal of variability has been observed in lead exposure data collected at various former ore-processing and urban sites, the structural equation models tailored to

each locale were similar to one another. These results occurred despite low levels of lead in blood at the ore-processing sites and obvious source and population differences. In conclusion, the technique of structural equation modeling is a powerful tool for helping resolve the complicated interactions between contamination in the environment and human uptake of these pollutants.

REFERENCES AND NOTES

1. Bornschein R. Bingham Creek Environmental and Human Health Lead and Arsenic Study. Final Report. Cincinnati, OH:University of Cincinnati, 1996.
2. Bornschein R. Butte-Silver Bow County Environmental Health Lead Study. Final Report. Cincinnati, OH:University of Cincinnati, 1992.
3. Bornschein RL, Succop P, Dietrich KN, Clark CS, Que Hee S, Hammond PB. The influence of social and environmental factors on dust lead, hand lead, and blood lead levels in young children. *Environ Res* 38:108-118 (1985).
4. Bornschein RL, Succop PA, Krafft KM, Clark CS, Peace B, Hammond PB. Exterior surface dust lead, interior house dust lead and childhood lead exposure in an urban environment. In: *Trace Substances in Environmental Health* (Hemphill DD, ed). Columbia, MO:University of Missouri, 1986:322-332.
5. Clark S, Bornschein R, Ryan J, Kochanowski A, Succop P, Peace B. The Cincinnati Soil-Lead Abatement Demonstration Project. In: *Lead and Soil: Issues and Guidelines*. Northwood, UK:Science Reviews, 1989.
6. Clark CS, Bornschein RL, Conway-Mundew L, Grote J, Menrath W, Pan W, Roda S. Urban Soil Lead Abatement Demonstration Project. Vol IV: Cincinnati Report. EPA/600/AP-93/00ID. Washington:U.S. Environmental Protection Agency, 1993.
7. Bornschein R. Urban Soil Lead Abatement Demonstration Project: Cincinnati Final Report. Cincinnati, OH:University of Cincinnati, 1993.
8. Bornschein R. Leadville/Lake County Environmental Health Study. Final Report. Cincinnati, OH:University of Cincinnati, 1997.
9. Bornschein R. Magna, UT Childhood Lead Exposure Study. Final Report. Cincinnati, OH:University of Cincinnati, 1997.
10. Bornschein R. Midvale Community Lead Study. Final Report. Cincinnati, OH:University of Cincinnati, 1990.
11. Bornschein R, Clark S, Pan W, Succop P. Midvale community lead study. *Chem Spec Bioavail* 3:149-162 (1991).
12. Bornschein R. Palmerton Lead Exposure Study. Final Report. Cincinnati, OH:University of Cincinnati, 1996.
13. Bornschein R. Sandy UT, Sources and Pathways of Lead Exposure in Sandy (Old Town), UT. Final Report. Cincinnati, OH:University of Cincinnati, 1998.
14. Bornschein R. Telluride Blood Lead Survey. Final Report. Cincinnati, OH:University of Cincinnati, 1987.
15. Bornschein R, Clark C, Grote J, Peace B, Roda S, Succop P. Soil lead-blood lead relationships in a former lead mining town. In: *Lead and Soil: Issues and Guidelines*. Northwood, UK:Science Reviews, 1989.
16. Bornschein R. Trail Childhood Lead Exposure Pathway Modelling. Final Report. Cincinnati, OH:University of Cincinnati, 1992.
17. Bornschein R. Aspen Environmental Health Study. Final Report. Cincinnati, OH:University of Cincinnati, 1997.
18. Morbidity and Mortality Weekly Report Update: Blood lead levels - United States 1991-1994. *Morb Mortal Wkly Rep* 46:141-146 (1997).